REMARKS

Favorable reconsideration of the subject application is respectfully requested in view of the above amendments and the following remarks. Prior to the present amendment, claims 30 and 32 were pending and under consideration. By the present amendment, new claims 33 and 34 are added to more specifically recite particular embodiments of the present invention. Support for this amendment is provided throughout the specification as originally filed, including, *e.g.*, on page 20, line 8 - page 21, line 7, page 9, lines 16-22, and page 20, lines 8-19. Accordingly, no new matter is added. This amendment is not to be construed as acquiescence to any rejection and is made without prejudice to prosecution of any subject matter modified by the amendment in a related divisional, continuation, or continuation-in-part application.

Rejection Under 35 U.S.C. § 102(e)

Claims 30 and 32 remain rejected under 35 U.S.C. § 102(e) as allegedly anticipated by U.S. Patent No. 5,622,852 (the '852 patent). More specifically, the Examiner asserts that the '852 patent teaches a monoclonal antibody to a mouse Bad polypeptide that would inherently bind to the human Bad polypeptide of SEQ ID NO:2, since the mouse Bad polypeptide is approximately 75% similar to the human Bad polypeptide. Furthermore, the Examiner asserts that the '852 patent teaches monoclonal antibodies directed to the BH1 domain of a mouse Bad polypeptide, which would also inherently bind to the BH1 domain of a human Bad polypeptide, given the degree of sequence conservation between the described mouse and Bad polypeptides.

In response to Applicants' arguments that antibodies to mouse Bad would not necessarily bind human Bad, particularly given the relatively low degree of primary sequence conservation and resultant differences in their secondary structure, the Examiner asserts that the claimed antibodies are not limited to antibodies that bind human Bad or its carboxy terminus in their native three-dimensional structure, but, rather, also encompass antibodies that bind to denatured human Bad, *e.g.*, as shown in SDS-PAGE, wherein epitopes are readily exposed. Based upon this remark, the Examiner concludes that the claimed antibodies are the same as those taught in the '852 patent.

Applicants respectfully traverse this basis of rejection and maintain that the Examiner has failed to establish a *prima facie* case of anticipation of the presently claimed invention by the '852 patent, since the '852 patent does not described antibodies that specifically bind to a human Bad polypeptide, and the Examiner has failed to demonstrate that the antibodies described in the '852 (which are specific for a mouse Bad polypeptide) would inherently bind to a human Bad polypeptide. As discussed extensively in the previous Amendment filed September 26, 2004, the Examiner bears the initial burden of establishing that the subject matter of the presently claimed invention is inherently present in the'852 patent. Furthermore, in relying on a theory of inherency, "the examiner must provide a basis in fact and/or technical reasoning to reasonably support the determination that the allegedly inherent characteristic necessarily flows from the teachings of the applied prior art." *Ex parte Levy*, 17 USPQ2d 1461, 1464 (BPAI 1990) (emphasis in original). Applicants maintain the position that in the instant case, the Examiner has provided no basis for concluding that the presently claimed subject matter is *necessarily* coextensive in scope with any antibody disclosed in the '852 patent. Accordingly, Applicants submit that the PTO has failed to meet its burden of establishing that the subject matter of the presently claimed invention is inherently present in the '852 patent.

With regard to the Examiner's assertion that the claimed monoclonal antibodies to human Bad encompass antibodies that bind to an epitope of human Bad that is masked in its native conformation but revealed upon denaturation, Applicants submit that the Examiner has failed to provide any reasoning as to how this is relevant to this basis of rejection or how such antibodies are necessarily inherently anticipated by the general description in the '852 patent of antibodies that bind mouse Bad. Whether in their native conformations or denatured, the human Bad and mouse Bad polypeptide sequences still share only 75% homology, which is clearly not sufficient to conclude that antibodies directed against one would necessarily cross-react with the other. Since this is the showing that the Examiner must make in order to establish a *prima facie* case of anticipation based upon a theory of inherency, Applicants submit that the Examiner has simply not met her burden, and, thus, has not demonstrated anticipation of the claimed subject matter by the '852 patent.

Furthermore, without acquiescence to this basis of rejection, Applicants have added new claims 33 and 34, which specifically recite the functional features that the claimed antibodies either interfere with the binding of human Bad to Bcl-2 or Bcl-X_L (claim 33) or inhibit apoptosis (claim 34). Applicants submit that these functional properties are only relevant to human Bad in its native conformation and not when inactivated by denaturation in SDS-PAGE. Accordingly, the Examiner's remarks related to antibodies that bind denatured Bad do not apply to these claims.

In light of the above remarks and amendments, Applicants submit that the presently claimed invention is clearly not anticipated by the '852 patent, and respectfully request that the Examiner reconsider and withdraw this basis of rejection.

The Director is authorized to charge any additional fees due by way of this Amendment, or credit any overpayment, to our Deposit Account No. 19-1090.

Applicants submit that the claims remaining in the application are now clearly allowable. Favorable consideration and a Notice of Allowance are earnestly solicited.

Respectfully submitted,

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